ALLYN O. KREPS (State Bar No. 29256)
RUSSELL G. WEISS (State Bar No. 173242)
LEWIS, D'AMATO, BRISBOIS & BISGAARD
221 North Figueroa Street
Suite 1200
Los Angeles, California 90012
(213) 250-1800

Attorneys for Petitioner
MICHAEL JACKSON

Of Counsel:
ROBERT S. DRAPER (State Bar No. 41832)
O'MELVENY & MYERS
400 South Hope Street
Los Angeles, California 90071-2899
(213) 669-6000

IN THE UNITED STATES DISTRICT COURT

CENTRAL DISTRICT OF CALIFORNIA

MICHAEL JACKSON, Petitioner, v. ARTHUR CALDERON, Warden of California State Prison at San Quentin, Respondent.	Case No. CV 91-4249-R **[DEATH PENALTY]** NARRATIVE STATEMENT OF DIRECT TESTIMONY OF ORM ANILINE, M.D., Ph.D. Date: January 24, 1996 Time: 9:00 a.m. Place: Courtroom 8

1. I am Orm Aniline, M.D., Ph.D., and this is the narrative statement of my direct testimony as an expert

witness called by petitioner, Michael Jackson, to render the following opinion:

> AT THE TIME OF THE HOMICIDE, MICHAEL JACKSON WAS IN A PCP-INDUCED AMNESTIC AND ANESTHETIZED CEREBRAL CONDITION WHICH, TO A 95% MEDICAL CERTAINTY, RENDERED HIM PHYSIOLOGICALLY INCAPABLE OF PREMEDITATING, DELIBERATING, OR FORMING AN INTENT TO KILL OFFICER WREDE.

2. My opinion is the result of three principal factors. The first is my education, training, and clinical experience. The second is scientifically established general properties and physiological effects of phencyclidine (PCP). The third is the actual clinical diagnosis and evaluation of Mr. Jackson's mental state at the time of the homicide. My testimony is generally divided into these three parts, with the detailed and specific opinion set out in the conclusion.

I.

EDUCATION, TRAINING AND CLINICAL EXPERIENCE

3. After graduating with a B.A. from Coe College, Cedar Rapid [sic], Iowa, in 1964, I went to graduate school at Yale University and received an M.S. in Chemistry and a Ph.D. in Physical Organic Chemistry from Yale in 1968.

4. I worked as a chemist and as a consultant for Dow Chemical Company and IIT Research Institute from

1968 to 1971. At Dow and IIT, I worked on the preparation, purification, and quantification of "Dioxin" – a substance that causes a myriad of human disorders, including cancer. I then taught chemistry and physics at Mercy College in Detroit for the academic year 1971-1972 and continued to work on the preparation and analysis of chlorodioxins, the "toxic" impurities found in Agent Orange.

5. I started medical school in 1972 and I received a M.D. from Wayne State University in Detroit, Michigan IN 1976. While at Wayne State University. I worked as an assistant to Dr. E. Domino, whose publication on the pharmacological properties of PCP lead to the development of Sernyl. Sernyl is the trade name for PCP and was used by surgeons to perform operations on humans without concern of cardiac or respiratory dysfunction.

6. After I received my M.D., I moved to Los Angeles and I did my Internship and Psychiatric Residency at the Los Angeles County Medical Center, Los Angeles, California, from 1976 to 1979.

7. I obtained my California Medical License in 1977 (No. G034463) and have maintained it to the present. I have been a Diplomate of the American Board of Psychiatry and Neurology since 1981.

8. I also hold a Controlled Substances Registration with the United States Drug Enforcement Administration (DEA) (No. AA9738773).

9. Since 1964, I have been a member of many professional organizations and associations, including the

American Chemical Society, the ACS-Division of Chemistry and the Law, the American Medical Association, and the American Academy of Psychiatrists in Alcoholism and Addictions.

10. Since 1971, I have received a number of academic appointments, including Assistant Professor of Chemistry and Physics at Mercy College of Detroit (1971-1972); Lecturer and Program Consultant for the Physicians Assistant Program at Mercy College of Detroit (1974-1976); Lecturer and Consultant at the USC School of Medicine (1977-1979); Assistant Professor of Psychiatry and Behavioral Sciences at the USC School of Medicine (1979-1984); and Assistant Clinical Professor of Psychiatry and Behavioral Sciences at the UC Irvine School of Medicine (1984-to date).

11. In addition, I have had a number of clinical appointments since 1979, including Staff Psychiatrist and Ward Chief at the Los Angeles County Medical Center (1979-1984); Staff Psychiatrist at the Long Beach VA Medical Center (1984-1993); and Community Mental Health Psychiatrist at the Orange County HCA (1985-to date). Through my clinical experience, I have treated over 1000 patients for PCP intoxication. Each of these patients tested positive for PCP and were tested to ascertain their PCP blood and/or urine level. At least 100 of these patients were grossly intoxicated with PCP. These grossly intoxicated patients had PCP blood levels of 100 ng/ml or greater.

12. I have received several academic honors and awards which include, but are not limited to, the Sandoz Award from the USC Department of Psychiatry in 1979,

and the Faculty Research Award from the USC Department of Psychiatry in 1982. Furthermore, I was a Fellow in Chemistry at Yale University from 1964 to 1968, and a Falk Fellow with the American Psychiatric Association from 1977 to 1978.

13. I have provided consulting services to community-based organizations as well as government agencies in the field of drug abuse and treatment.

14. I have published a number of articles in the field of drug abuse and treatment, including several articles on Phencyclidine. The following is a list of some of the articles in peer review journals that I have authored on the subject of PCP:

(a) *Capillary GC-Nitrogen Detector Measurement of Phencyclidine (PCP) and Other Arylcycloalkylamines in the Picogram Range*, J. Chromatography (1980);

(b) *Incidental Intoxication with Phencyclidine*, J. Clin. Psychiatry (1980);

(c) *The Urban Epidemic of Phencyclidine Use; Laboratory Evidence from a Public Psychiatric Hospital Inpatient Service*, Biol. Psychiat. (1980);

(d) *Phencyclidine (PCP): A Review and Perspectives*, Crit. Rev. in Toxicol. (1982);

(e) *The Dilemma of the Toxic Psychosis: Differential Diagnosis and the PCP Psychosis*, Psychiatric Annals (1982);

(f) *Occupational Intoxication and Long-Term Persistence of Phencyclidine (PCP) in Law Enforcement Personnel*, Clin. Toxicol. (1982);

(g) *Phencyclidine Malignant Syndrome: A Case Report,* Arch. Neurol. (1982); and

(h) *Most PCP Intoxication Is Missed,* Continuing Education for the Family Physician (1983).

15. I also have published a number of books and chapters in books in the field of drug abuse and treatment including several articles on PCP. The following are two of the books and chapters in books that I have authored on the subject of PCP: Aniline, O. & Simpson, G., "Psychiatric Aspects of Phencyclidine", in *Advances in Neurotoxicology,* (L. Manzo ed.), Pergamon Press, Ltd., Oxford, England (1980), and Aniline, O., Allen, R.E. & Pitts, F.N. Jr., "Clinical and Kinetic Aspects of Phencyclidine (PCP)", in *PCP (Phencyclidine): Historical and Current Perspectives,* (Domino, E.F. ed.), NPP Books, Ann Arbor, Michigan (1981).

16. I have given presentations at professional and academic conferences in the field of drug abuse and treatment, including the role of psychiatrists in capital-murder cases and capital post-conviction proceedings.

17. I have been in private practice since 1979 and have specialized in consulting and forensic psychiatry. During this time, I have participated in over 300 cases, including 75 criminal cases. I have testified in approximately 50 homicide cases, and have been involved in six death penalty appeal cases. I have extensive experience in interviewing criminal defendants and witnesses. I also have reviewed transcripts of witness statements and testimony, transcripts of grand jury proceedings, and police

reports. I have worked with attorneys in trial preparation, including capital cases, and I have been involved in providing expert evidence in capital cases since 1981.

18. I have testified and been qualified as an expert witness in a number of criminal and civil cases brought in state and federal courts in California.

19. My expertise in the area of forensic psychiatry encompasses drug/alcohol abuse and drug/alcohol treatment. In particular, I have testified extensively on both the beneficial and adverse effects that drugs and medication have on an individual's mental state and behavior.

20. My attached curriculum vitae accurately discloses my credentials, including degrees, current position, experience, expertise, and in part, publications regarding PCP, and other personal data. A true and correct copy of my curriculum vitae is attached hereto as Exhibit "A".

II.

PHENCYCLIDINE – GENERAL PROPERTIES AND PHYSIOLOGICAL EFFECTS

21. Phencyclidine ("PCP") was originally developed in the 1950's as a general anesthetic. Initially tested on humans, it was unique in inducing a catatonia-like state which permitted performance of minor surgical procedures. "The overall behavioral state (patients exhibited during PCP-induced anesthesia] was one of evident and marked dissociation from the environment without complete loss of consciousness." ("Recent Advances in the Phencyclidine Model of Schizophrenia", Daniel C. Javitt,

M.D., Ph.D., and Stephen R. Zukin, M.D., 148 American Journal of Psychiatry, 1301-1308 (1991) at 1302.) PCP was withdrawn from clinical use in 1965 because of the psychotic states it induced in patients, which states lasted typically for 12 to 72 hours but were also known to last for up to 7 to 10 days. (*Ibid.*)

22. PCP has three basic properties: anesthetic, stimulant, and psychotomimetic.

23. The human brain has what are known as PCP-specific receptors. Receptors are the portion of a cell which "receives" or absorbs certain elements or chemicals from the bloodstream. PCP-specific receptors are present in the brain cells most impaired by PCP. PCP-specific receptors are called NMDA (N-methyl-D-aspartate) receptors in the scientific community. These receptors govern synaptic plasticity, long term potentiation, learning and memory, and developmental plasticity.

24. NMDA receptors are involved in the fine control of coordinated movement, behavioral patterns, and memory and learning. These receptors are also involved in several pathological states, including epileptic phenomena and neurodegeneration, caused by prolonged depolarization of cells by endogenous and exogenous amino acids. Stated differently, NMDA receptors affect cortical cell physiology. They are located in cells of the frontal cortex and hippocampus, areas required for learning and memory. When inhibited or blocked by PCP, these cells do not function.

25. Research on the NMDA receptors shows that once the receptors are blocked, an individual becomes amnestic: The person is impaired in the ability to think

while intoxicated and to recall events occurring during the intoxication.

26. PCP diminishes the perception of pain and prevents the intoxicated individual from experiencing pain. However, unlike alcohol and other general, non-specific drugs with anesthetic characteristics that impair both the brain and the brain stem, PCP primarily affects the cortex, the part of the brian [sic] which processes cognitive thought, including premeditation, deliberation and the formulation of intent.

27. Early clinical use of PCP as an anesthetic significantly reduced the risk of cardiac arrest, hypertension and respiratory depression, as it does not impair the brain stem – the body's "computer" for all involuntary or reflexive muscles such as the heart. The brain stem – which also processes performance of repetitive or reflexive actions – is generally unaffected by PCP. Individuals under the influence of the drug are known to be capable of performing reflexive tasks for this reason. The retrieval of old, familiar information does not require an intact memory system.

III.

DIAGNOSIS AND EVALUATION OF CEREBRAL CONDITION OF MICHAEL JACKSON AT THE TIME OF THE HOMICIDE, AUGUST 31, 1983

28. In making my diagnosis and evaluation of the cerebral dysfunction of Michael Jackson at the time of the homicide, I have taken the basic and medically pertinent facts of the incident from the trial transcript, the Amended Petition for Writ of Habeas Corpus and related

documents, the medical and other records produced in discovery, the 1995 PET Scan, and scientific and medical literature in 1983 and to date.

A. The Events Of August 31, 1983

29. Michael Jackson had a chronic history of PCP and polysubstance use since age 13. Michael Jackson was unable to work at a regular job. He smoked PCP on an almost daily basis and supplemented his income by helping his friend, James Butler, sell PCP.

30. Michael spent the night of August 30, 1983 with friends, drinking alcohol and taking cocaine and amphetamines that some of his friends wanted him to sell. After sleeping a couple of hours, Michael phoned Butler to ask if Butler had any odd jobs for him. Told he could wash Butler's walls for money, Michael asked Butler to pick him up.

31. After they arrived at Butler's house, Butler told Michael he didn't have all the money to pay Michael for the work but that he had some "Super Kools" – PCP-laced cigarettes – as partial payment. Michael smoked one of the PCP-laced cigarettes. Then Michael and Butler were joined by two women. Michael shared two or three additional Super Kools that Butler had prepared by dipping them into the PCP he stored in a refrigerator located in his garage. Shortly after smoking the Super Kools, Michael jumped up, kicked off his shoes and darted out of the garage.

32. Sometime thereafter, between about 11:50 a.m. and noon, Michael was observed by a witness, "jumping

on cement and he was yelling and just running around." "He was just yelling. – he wasn't saying no words." He was observed falling down, jumping up, and then diving head first or doing "swan dives" into the street.

33. Shortly after noon, he was observed on Francisquito staggering down the sidewalk. At one point, he was observed ducking down, squatting in some shrubs. Michael was in a disheveled condition, shirt-tail out, and wearing socks but no shoes[.] This is blatantly bizarre behavior which is associated with PCP intoxication.

34. Between about 12:15 and 12:17 p.m., Michael was observed at Francisquito and Lark Ellen, in the intersection, appearing "like he was disorientated, not knowing which way to go." He started to cross Lark Ellen, only to change his mind repeatedly, finally heading back east on Francisquito, toward Glenview. He was pulling and slapping at his hair as if he was "drunk or high or something . . . "

35. Officer Wrede was at a nearby street. A passing motorist, concerned that Michael might hurt himself, stopped and reported to Wrede that "there was a man on the corner at Lark Ellen and Francisquito acting very strangely and disorientated."

36. At about 12:20 p.m., a long-time friend of Michael's family was driving down Francisquito when she noticed Michael approaching the intersection of Francisquito and Glenview from the west. She drove up next to Michael, rolled down her window and hollered Michael's nickname, "Popeye", at him. When Michael didn't respond, she started driving on. As she did so, she looked into her rear view mirror and noticed that the seat

of Michael's trousers was dirty. She then made a U-turn, came back around, rolled down her window, and again hollered "Popeye" at Michael. Again, Michael did not respond, but kept on walking. Feeling concerned that something might be wrong, she went to get Michael's mother. Twice Michael Jackson had not responded to this person familiar to him – someone non-threatening and a family friend.

37. At about the same time, Officer Wrede radioed that he would be checking a possible Penal Code 647 violator (drunk or intoxicated) on Francisquito, east of Lark Ellen.

38. As he continued east on Francisquito, Michael was observed walking "kinda crooked" by a witness who believed him to be drunk or high.

39. At 12:23:04, Officer Wrede radioed that he would be out with the "possibly dusted" suspect, and requested the assistance of additional officers. Officer Wrede then parked his car, with its lights flashing, at the intersection.

40. Wrede, walkie-talkie in hand, exited his car and walked around it to encounter Michael, who was standing on the sidewalk. He called to Michael, "Where are you going?" Michael responded by pointing north along Glenview. Although Wrede asked Michael to sit on the curb, Michael remained standing. At this time, 12:23:59 p.m., Wrede used his walkie-talkie to ask where his back-up assistance was coming from. Wrede again asked Michael to sit on the curb after which Michael turned and headed north on Glenview. In effect Michael Jackson disregarded or failed to respond to a uniformed officer.

Michael Jackson was unresponsive to a friend and to an official. He was not responding to people in a normal manner.

41. Wrede then took out his baton and struck Michael behind the knees. This blow was intended to illicit a painful response. Michael Jackson was not disabled by pain; instead he was provoked and swung wildly at Wrede falling down as he did so. The two of them wrestled and fell. Wrede kicked Michael Jackson away. Wrede repeatedly sprayed Mace into Michael's face, without apparent effect. Chemical deterrents, like Mace, are often ineffective on "dusted" individuals.

42. It was 12:25:19 p.m. when Wrede reported Michael was getting a stick and again asked for back-up. Michael attempted to pull up a nearby tree sapling, eventually pulling up a six-foot support stake. He waved and swung the stake at Wrede several times before he threw it at Wrede, who dodged it.

43. The two continued to struggle. As Michael threw some punches, Wrede used his baton to strike several blows to Michael's stomach, with no apparent disabling effect to Michael Jackson. Wrede disengaged and ran to his police car, reaching across the front seat from the driver's side for his car radio or shotgun. Michael followed Wrede but to the passenger side, and also reached across the front seat toward the shotgun. The two wrestled for control of the shotgun which was secured in its rack. At 12:26:16 p.m., Wrede reported that Michael had grabbed his shotgun and rack.

44. At 12:26:28 p.m., Michael pulled the 12 gauge shotgun, rack and all, from the car. He then began running his hands up and down the rack, as if trying to cock the shotgun. Wrede got out of the car and took cover behind the left rear of the vehicle, service revolver drawn.

45. Michael, on the passenger side of the car and in front of the flashing light bar, pointed the shotgun over the roof in the direction of the light bar and Wrede. Wrede was still at the left rear of the car, not more than 6 to 8 feet from Michael, his revolver aimed toward Michael. Michael fired a single nine-pellet shot. One bent pellet struck Wrede in the eye, entered his brain, and killed him.

46. Michael Jackson did not go to see if Wrede was hurt. After firing the single shot, Michael Jackson just began stumbling west on Francisquito, the shotgun still in the rack in his left hand. The entire incident had taken less than four minutes and the actual physical encounter less than 2 1/2 minutes.

47. At 12:27:13 p.m., when the report of an officer down was dispatched, several officers with a police dog had just arrived at the scene in response to Wrede's earlier request for back-up. One of the officers screamed to the others to shoot Michael. In response, Michael reportedly yelled that he was "going to kill you." The police dog was then released, and the dog and Michael circled each other. Michael struck at the dog with the gun. He stunned the dog, but it still managed to bite him on the leg, causing him to drop the shotgun, but it did not cause him to stop fighting.

48. Michael was gang-tackled by several officers, who subdued him with fists and batons as the police dog bit him several times in the groin. At 12:31:29 p.m., Michael was reported to be in custody and police obtained his name and address from witnesses at the scene.

49. After Michael was taken to the West Covina jail, paramedics were called at 1:00 p.m. because of Michael's physical condition. When the paramedics arrived at 1:10 p.m., they recorded a pulse of 152/min; respirations at 60/min. and a blood pressure ("Bp") of 90/40. No intravenous line ("IV") was established but Michael was put into a "mast suit" and transported to the Queen of the Valley Emergency Room, where he arrived at 1:40 p.m.

50. Michael Jackson was so physiologically impaired that he was unable to respond to verbal commands. At 1:40-1:45 p.m., the "mast-suit", placed by paramedics to sustain blood pressure and blood flow to the brain, was removed. An EKG recorded at 1:46 p.m. was grossly abnormal and showed a fast heart rate. At 1:45 p.m., an IV was started.

51. Minutes after the IV fluids were infused, a blood sample was taken that later was found to contain 176 ng/ml of PCP. Depending on the length of time after the IV began until the blood sample was taken, the PCP would have been higher than reported, since the IV would have diluted the PCP in the blood and would have produced a lower number. At 1:50 p.m., Michael Jackson was mumbling unintelligibly. This indicates that he had an impaired level of consciousness. At 2:10 p.m., a urine sample was taken which was qualitatively positive for

PCP. The urine sample later revealed the presence of cocaine, alcohol and amphetamine-like drugs.

52. After admission to Queen of the Valley Emergency Room and for hours afterward hospital notes indicate that Michael Jackson was in shock, incoherent and semi-conscious. A blood gas sample showed severe acidosis (pH of 7.13), which certainly helped to precipitate renal (kidney) failure from rhabdomyolysis. This is a condition found in PCP intoxicants. At 2:50 p.m., a police photographer took color identification photographs which showed a battered man, strapped to a bed and naked to expose leg and groin wounds. (Exh. A to Petition) Michael was then transferred to Los Angeles County Medical Center, LAC/USC.

53. The medical record at LAC/USC indicated that Michael Jackson was admitted at 1700 (5:00 p.m.). The admitting doctor reviewed the records from Queen of the Valley. At 2200 (10:00 p.m.) a surgical resident noted inappropriate speech and behavior and subconjunctival hematoma (swollen left eye) causing blurred vision. He was started on dialysis after an intravenous pyelogram showed damage to both kidneys and he remained in restraints.

B. Diagnosis and Evaluation

54. The foregoing recitation of events are the most significant facts which underlie my diagnosis and evaluation of Michael Jackson's cerebral dysfunction at the time of the homicide on August 31, 1983. In marking this diagnosis and evaluation I am going to show both how

this could have been done to a reasonable medical certainty of 90% in 1983 and why my opinion today is of a 95% medical certainty.

55. PCP and its closely related cogener, Ketamine, have served as models for the study of amnesia produced in medical procedures such as surgical anesthesia. Levels of PCP in excess of 100 ng/ml produce amnestic states such as those utilized during surgical anesthesia. Ketamine is a dissociative anesthetic which has been in use for over 25 years and has been studied in many clinical situations. A dissociative anesthetic has its primary effects on the brain's cortex. Thought occurs in cortical tissue as opposed to the brain stem where most anesthetics have their impact on consciousness, heart rate and respiration. Individuals given Ketamine anesthesia may be able to follow simple directives during their surgery but have no long term memory of the procedure. This phenomenon is well recognized by clinicians experienced with the use of dissociative anesthetics. In parallel fashion, Michael Jackson's mental state at the time of the incident and his inability to recall the event may be explained by the anesthetic properties of PCP.

56. Scientific advances in the pharmacology of PCP permit a more certain correlation to be drawn between high levels of PCP in the blood and brain cell impairment. The brain cells most impaired by PCP have PCP specific receptors. These receptors regulate cell physiology and control the mental processes of learning and memory. When inhibited by PCP, these cells do not function. Learning and memory storage do not take place, just as occurred with Michael Jackson during the homicide. When these cells are damaged or die from excessive drug

use, the individual's ability to acquire, process and retrieve information is permanently damaged. Some of these findings have been elucidated and documented by physician/scientists utilizing Positron Emission Tomography ("PET") scans and Evoked Potential methodology.

57. When a person uses alcohol in conjunction with cocaine, the liver combines and chemically transforms these drugs into a chemical compound known as cocaethylene. Although cocaethylene could not have been detected by the standard toxological screens used by hospitals in 1983, my diagnosis is that Jackson probably was under some influence of it at the time of the incident. On the night before Michael consumed alcohol, snorted cocaine, and smoked marijuana. Furthermore, Michael's urinalysis which was obtained shortly after the incident shows that he was intoxicated with amphetamines, alcohol and cocaine. Apparently, these results were ignored in 1983 because of, or overshadowed by, the extremely high levels of PCP found in Jackson.

58. Chronic use of cocaine leads to brain tissue damage. The damage can now be demonstrated by imaging techniques such as PET scans. Recent work shows that the areas compromised by cocaine and by inference cocaethylene can be visualized by these imaging techniques. The areas of concern include the inferior prefrontal cortex and regions of the temporal lobes – areas known to regulate mood and cognitive integration. Similar deficits have been demonstrated in amphetamine users. Cocaethylene is a very potent and very lethal drug twenty times more powerful than pure cocaine. Cocaethylene is as potent as cocaine in its euphoric effects. Cocaethylene is more fat soluble than cocaine and

tends to build deposits in fat tissues such as the brain. Cocaethylene's effects on cognition and behavior are similar to those attributed to PCP. I believe that cocaethylene, in combination with the PCP and amphetamines severely impaired Jacksons' judgment, cognition and memory.

59. Since the time of petitioner's trial, scientific knowledge has come to support the conclusion that a PCP blood level of 176 ng/ml would definitely represent a level sufficient to produce surgical anesthesia, and cause gross impairment of a person's ability to perceive his situation and to recall that situation at a later time, regardless of one's tolerance to the motoric effects of the drug. This conclusion is based partly on the fact that 176 ng/ml maintains a high PCP blood level for hours, and partly on the fact that, in my personal experience treating hundreds of persons intoxicated with PCP, I am unaware of a single person with a blood level above 100 ng/ml who was not grossly impaired – physiologically and/or psychiatrically. A chart indicating the physiological effects of PCP is attached hereto as Exhibit "B". At the time of the homicide, Jackson had a PCP blood level of 176 ng/ml and was physiologically incapable of processing information in normative fashion. Jackson was grossly impaired to the point where I believe he did not know what he was doing and could not distinguish that he was firing a gun at a police officer. Since Officer Wrede was only 6-8 feet away from Jackson, a shogun blast aimed at him by Michael would have been devastating, rather than resulting in one of nine pellets ricocheting grotesquely into Officer Wrede's eye.

60. At the time of Jackson's trial in 1984, it was not known to a scientific certainty that humans had PCP

specific receptors. One of the more comprehensive articles detailing new developments in the PCP field is Javitt & Zukin, *Recent Advances in the Phencyclidine Model of Schizophrenia*, 148 American Journal of Psychiatry 1301 (1991. (Exh. C). These PCP specific receptor, called NMDA receptors, are named after the substance N-methyl-D-aspartate. NMDA receptors govern synaptic plasticity, long-term potentiation, learning and memory, and developmental plasticity. NMDA receptors are involved with the fine control of coordinated movement, behavioral patterns and memory and learning. They are also involved in several pathological states including epileptic phenomena and neurodegeneration caused by prolonged depolarization of cells by endogenous and exogenous amino acids. *The NMDA Receptor*, J.C. Watkins & G.L. Collingridge, eds., 14 (1991).

61. Put another way, PCP receptors affect cortical cell physiology. NMDA receptors are located in cells of the frontal cortex and hippocampus, areas required for learning and memory. Dr. Zukin's studies have shown that PCP levels above 100 ng/ml would block these receptors and saturate the NMDA receptors of cortical cells. Research on the PCP receptors shows that once these receptors are blocked, the affected individual becomes amnestic. He is impaired both in his ability to think at the time of intoxication and in his ability to recall the events of that time. This is why Jackson, with a PCP level of 176 ng/ml was unable to think at the time of the homicide and unable to recall his actions at a later time. His brain was physiologically incapacitated; it simply could not function at that level.

62. PCP does not affect the brain stem which also processes performance of repetitive or reflexive actions. It is, therefore, not surprising that Jackson could fight, run, perform other reflexive tasks and even fire the gun, yet have no actual consciousness or recollection of having done so. Similarly he could give his name, telephone and other dates and, yet, not be able to retrieve information acquired only momentarily before hand. The retrieval of old, familiar information does not require an intact memory system. Demented individuals commonly recall old information but cannot process the unfamiliar. In fact, as a drug of abuse, PCP has been used in recreational doses by individuals performing monotonous, strenuous work.

63. The medical records which I reviewed on petitioner following the incident show that petitioner was obtunded, non-responsive and still in an anesthetized state. Hospital staff were unable to obtain even rudimentary information from petitioner until several hours after his admittance. This also supports Jackson's statements of amnesia, since it is recognized transient cognitive and psychomotor impairment following anesthesia and surgery is normal. Partial awareness or recall of the surgical experience occurs in about 1% of surgical patients.

64. The medical records also show that petitioner was placed on dialysis hours after being admitted to LAC/USC. Dialysis is required in situations where muscle cells have broken down and coat the kidney, causing kidney failure. This phenomenon, called rhabdomyolysis, can be caused by severe PCP intoxication, as well as, by physical trauma. As reflected by the medical records, the

blood test results, and the photographs of Jackson showing his physical state and wounds, both were present here.

65. Dialysis is a serious procedure which may impair an individual's cognitive ability to conduct focused mental testing processes. Someone who has recently been on dialysis may be cognitively impaired and able to demonstrate learned or familiar knowledge. The acquisition of new information requires an intact cortical system. An impaired system will demonstrate primitive or rudimentary thought not consistent with the cognitive requirements involved with premeditation, reasoning, and deliberation.

66. My testimony here is all directed to my expert opinion that Michael was physiologically incapable of deliberating, premeditating or formulating an intent to kill Officer Wrede at the time of the homicide on August 31, 1983. However, and by coincidence, I was actually a defense witness at the guilt phase of Jackson's trial in 1984 and I testified as an expert on the general effects of PCP intoxication. It is not my purpose to repeat that testimony, but I must comment that there was sufficient medical evidence and experience then available or that could have been available, for me to have given essentially the same opinion I am testifying here.

67. In 1983, I was a full time psychiatrist at Los Angeles County Medical Center, the same county facility which treated Jackson. During that period of time the hospital would routinely treat patients who were admitted while believed to be under the influence of alcohol or

some other drug. These patients would be held for observation until they were able to cognate effectively and behave safely. My department was responsible for making that determination. The determination would be made by evaluating the patient serially during their hospitalization. Upon admittance, blood and urinalysis tests, and often an electroencephalogram ("EEG") test, would be conducted.[1] These tests provided a reference point for the specific patient in addition to helping determine if the condition was drug-related. Concurrently, the patient would be given various psychological-neurological tests which would be considered in conjunction with the other tests results to determine the mental condition of the patient, namely, was there an impairment, what treatment was needed, and could the patient be rehabilitated? All of these tests would he repeated regularly, on a daily or weekly basis, to determine the remaining drug level and the change in the mental state. The patient would be monitored until the drug levels in his body disappeared or dropped to low levels such that he was able to perform the most basic mental processes and could function safely in the community.

68. The psychological-neurological tests include (a) MMPI, and associated tests of mental function; (b) computerized neuropsychological tests, *i.e.*, psychological tests which permit specific tests of concrete and abstract mental tasks; and (c) the "EEG and Evoked Potentials,"

---

[1] The EEG test is a well-recognized test often used to help physicians determine the etiology of a patient's condition. It was used in 1983 to help treat patients who were disoriented, violent or who experienced seizures.

which measure involuntary brain wave responses to light and sound.

69. All of the above-described tests were available and commonly used at Los Angeles County Medical Center in 1983. However, with the exception of initial blood and urinalysis tests, none of the tests were conducted, even though it appears that police suspected petitioner was a "duster" who was intoxicated at the time of the homicide. It also appears that hospital personnel likewise suspected PCP intoxication. While Jackson's intoxication was confirmed by a blood test and urinalysis, the status of his cognitive abilities objectively could have been determined further by repeated blood tests and urinalysis combined with one or more of the described psychological-neurological tests, as was commonly done when psychiatric or neurological evaluations were performed at LAC/USC. Given the circumstances surrounding Jackson's admittance to the hospital, the killing of a police officer, obvious intoxication, his obtunded state, the impact of dialysis, and the need of the police to obtain a statement, Jackson's mental state could have been rigorously evaluated. Even if these tests had been conducted a month, maybe two, after the incident, the results would have provided objective support for Michael Jackson's amnestic state and his cognitive impairment.

70. These tests apparently were never conducted. When Michael was eventually evaluated by Drs. Mead and Skrdla, neither doctor attempted to determine what his mental state was at the time of the incident. Adipose tissue removal could have been performed which would have shown levels of PCP and possibly other drugs. Using this procedure, an incision is made into the patient

to obtain fat cells for analysis. Since PCP and many other drugs are fat soluble, they remain in the fat for several months or years. Used in conjunction with the battery of psychiatric tests, adipose tissue removal could have established, several months later and before trial, the validity of petitioner's amnestic condition at the time of the incident. Instead, both doctors focused only on whether Michael Jackson was then mentally able to understand the trial proceedings.

71. After as I was retained in this case in 1992, I was asked to conduct a clinical evaluation of Michael Jackson, who is incarcerated on death row at San Quentin. I visited him in the fall of 1992 for two consecutive days to obtain medical and factual background, to test his memory and veracity, and to make clinical evaluation of his present mental condition. I am of the opinion that Jackson was a chronic PCP user for at least 7 years before the homicide and used PCP most days of the week, I also am of the opinion that Jackson has no memory of the homicide in 1983 and has not recovered any memory since 1983. His PCP-induced cerebral dysfunction at the time of the homicide physiologically shut down his brain so that he was not conscious of what he was doing and his brain could not register memory of events between 11:30 a.m. and 1:30 p.m. on August 31, 1983.

72. I am also convinced based on my efforts to trick, to entrap, or to deceive Michael that he was telling me the truth to the best of his ability about the events of August 31, 1983. This is fortified by my discussions with all his attorneys in this case, Messrs. Allyn O. Kreps, Charles D. Ferrari, Molly S. Murphy, Russell G. Weiss, Rex S. Hungerford, as well as current co-counsel Robert S. Draper of

O'Melveny & Myers, each of whom similarly tested Michael's memory and veracity.

73. Based among other things, upon this evaluation, I advised the attorneys that a PET Scan could provide objective scientific evidence that showed current PCP brain damage. Since Jackson has been incarcerated since August 31, 1983 and was without any known access to PCP or other drugs, if the PET Scan showed current PCP brain damage, that would be further evidence of the physiological condition of his brain at the time of the homicide. The result of the PET Scan of Michael, finally obtained in October 1995, is set forth in the Narrative Statement of Direct Testimony of Joseph Chong-Sang Wu, M.D. Those results confirm and strengthen my opinion expressed here.

74. I also visited Michael Jackson in February 1995 to re-evaluate him and that evaluation reconfirmed and strengthened my opinion of his physiological condition at the time of the homicide.

IV.

CONCLUSION AND OPINION

75. The Diagnostic and Statistical Manual (DSM-IV) of the American Psychiatric Association describes PCP intoxication as:

> Specific signs and symptoms are dose related. Lower doses of phencyclidine produce vertigo, ataxia, nystagmus, mild hypertension, abnormal involuntary movements, slurred speech, nausea, weakness, slowed reaction times, euphoria or affective dulling, loquacity, and lack of concern.

> Disorganized thinking, changed body image and sensory perception, depersonalization, and feelings of unreality occur at intermediate doses. Higher doses produce amnesia and coma, with analgesia sufficient for surgery, and seizures with respiratory depression occur at the highest doses. Effects begin almost immediately after an intravenous or transpulmonary dose, reaching a peak within minutes. Peak effected occur about 2 hours after oral doses. In milder intoxications, the effects resolve after 8-20 hours, whereas signs and symptoms of severe intoxications may persist for several days. Phencyclidine-Induced Psychotic Disorder may persist for weeks.

76. Based upon my testimony here and using this standard, my expert opinion is that at the time of the homicide on August 31, 1983, Michael Jackson was in a PCP-induced amnestic and anesthetized cerebral condition which, to a 95% medical certainty, rendered him physiologically incapable of premeditating, deliberating, or forming an intent to kill Officer Wrede.

I declare under penalty of perjury that the foregoing is true and correct, and that this Narrative Statement of Direct Testimony was executed on the 9th day of January, of 1996, at Los Angeles, California.

/s/ Orm Aniline, M.D., Ph.D.
ORM ANILINE, M.D., Ph.D.